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Iran Red Crescent Med J. 2016 February;18(2): e22157. doi: 10.5812/ircmj.22157

Published online 2016 February 6. **Research Article**

Long-Term Exposure of Lead Acetate on Rabbit Renal Tissue

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Received 2014 August 8; **Revised** 2015 May 19; **Accepted** 2015 June 8.

Abstract

Background: Lead has been widely used in different industries for ages. It is one of the heavy metals, highly poisonous even at low doses, and has biochemical, physiological and behavioral side effects on human and animals. It has been shown that lead has toxic effects on different tissues such as neural and genitourinary tissues, cardiovascular systems and blood. Therefore, high attention has been paid to its environmental pollutions.

Objectives: Although many histological and biochemical studies have reported about the effects of lead on the renal tissue, there are a few studies about the ultrastructure and morphometric effects of lead on the kidney. Hence, the aim of this study was the evaluation of morphology and morphometrics of rabbit renal urinary barrier ultrastructure following long-term exposure to lead acetate.

Materials and Methods: In this experimental study, 20 male New Zealand rabbits were divided into control and test groups (10 in each). The test group was injected intraperitoneally with chronic dose (8.5 mg/kg of body weight) of lead acetate and for the control group the same volume of normal saline was used, every other day for 10 weeks. After anesthetizing, the biopsies of renal tissues were taken for light and electron microscopic morphometric and morphologic analyses.

Results: Long-term exposure to lead acetate caused histopathology effects including dilatation, congestion, nuclei heterochromatic effects, increase in diameter of renal tubules and urinary barrier thickness in rabbit renal tissue.

Conclusions: Quantitative and qualitative results of long-term lead acetate exposure showed many histopathology side-effects, especially in the urinary barrier.

Keywords: Kidney, Lead Acetate, Morphologic, Morphometric, Urinary Barrier, Glomeruli

1. Background

Lead has been extensively used in different industries for thousands of years. Relevance between lead poisoning and renal disease in humans has been recognized for more than a century. Lead is one of the most important air pollutants and poisonous even at low concentration. It has many side-effects including physiological, mental and histological disorders (1). Exposure to environmental hazardous doses of lead is one of the important health risk factors, particularly in at-risk human and animal populations. Although adults are vulnerable to lead poisoning, children and infants are more at risk due to their lower tolerance and immature immune systems (2). Some of the common lead pollution sources are water pipes made of lead, soldering wire, lead-based paintings, ceramic screens, food packages, pastry powder, leads painting sheets, agriculture products enriched by fertilizers, fungicides, herbicides and so on. Furthermore, lead may contaminate the body through inhaling dust or consuming plant products in polluted farms (3-5). Chronic exposure to heavy metals has adverse effects on humans, animals and plants. Moreover, lead poisoning can affect different systems including neural, cardiovascular and genitourinary (6). Therefore, a lot of attention has been paid to its environmental pollutions (7-9). Recent studies have indicated that it could cause many histochemistry and histopathology changes in renal tissue, including eosinophilic intranuclear inclusions in proximal tubular cells, consisting of lead-protein complexes, mitochondria swelling, and disturbances in proximal tubular function (10). These may lead to severe side-effects such as focal-glomerular sclerosis, glomerular hyalinization, vacuolization, tubular hyperplasia, tubular adenoma, necrosis, tubular dilation and nuclear picnosis (11). However, the severity of adverse effects depends on its plasma concentration and duration of exposure. Clinical signs emerging from renal lead poisoning have been well described

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